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[*From the Proceedings of the Physiological Society*, March 12, 1921.]
*Journal Physiology, Vol. LV.*

**Engelmann's evidence for the independence of excitability and contractility in heart muscle.** By T. Lewis and A. N. Drury[1].

In his paper "Ueber die bathmotropen Wirkungen der Herznerven," *Archiv f. Anat. u. Physiol.*, 1902, Physiol. Abth., Suppl. Bd. I.), Engelmann brings forward evidence to show that the two properties known as excitability and contractility are wholly independent functions of heart muscle. This, the main conclusion of his paper, has received a wide measure of acceptance; his most striking evidence consists of a series of four myograms (Figs. 9–12) from the frog's auricle. The auricle was stimulated rhythmically by shocks reaching it at a rate of one per

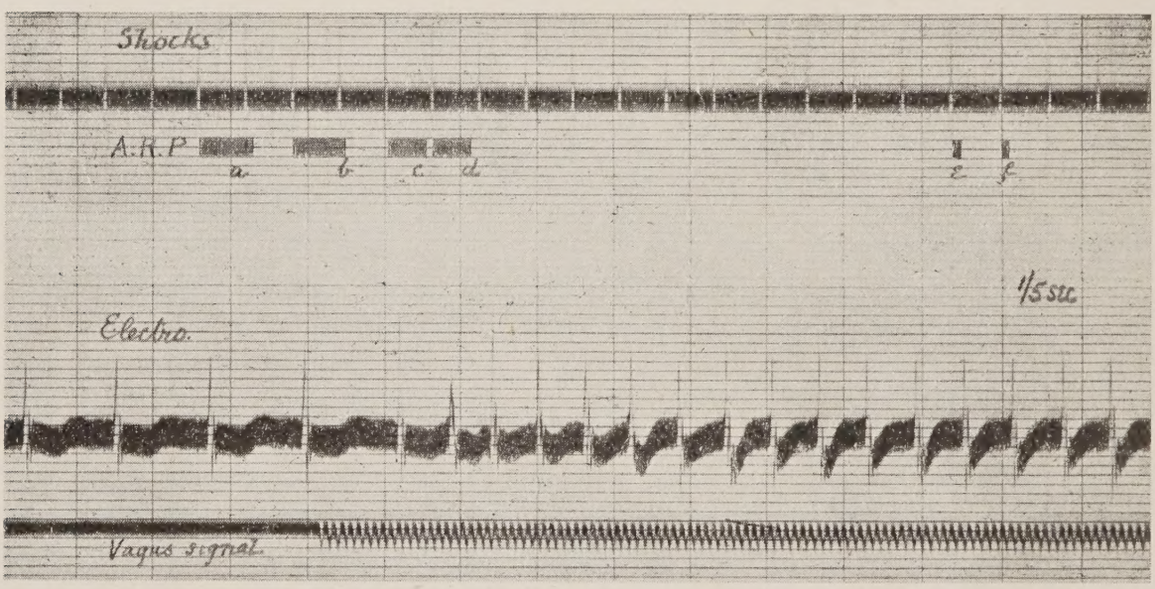


Fig. 1. The top record is of the shocks entering the auricle (rate 500 per minute), the middle record is an electrogram taken from the auricular muscle some distance from the point stimulated. A.R.P. = absolute refractory period represented diagrammatically. Before vagal stimulation the auricle is in a stable condition of half-response; shortly after and as a result of vagal stimulation it breaks into complete response. The shocks were well above threshold value throughout. The A.R.P., during the period of half response, exceeds slightly the intershock interval (as represented at *a* and *b*). At a late stage of vagal stimulation the A.R.P. is measured at about ·02 of a second (as represented at *e* and *f*). The appearance of complete response evidently results from shortening of the A.R.P. (as represented at *c* and *d*).

second; the auricle responded to alternate shocks only. The vagus was now stimulated reflexly, whereupon the strength of the auricular beats declined while the auricle responded to each stimulus reaching it. Engelmann interprets this change as due to a decline of contractility (negative-inotropic nerve-influence) with a rise of excitability (positive-bathmotropic nerve-influence).

[1] Working on behalf of the Medical Research Council.

The phenomenon witnessed by Engelmann has been recently investigated and its true explanation proves to be much more simple. When the auricle (in the case of our experiments, the dog's auricle) is driven at a sufficient speed, it fails to respond to alternate shocks; these shocks fall, as we are able to show, a little before the ending of the absolute refractory periods of the preceding systoles. If now the vagus is stimulated, the systoles of the auricle are not only reduced in amplitude but they are also shortened, the corresponding refractory periods are reduced (see *Proc. Physiol. Soc.*, Dec. 18, 1920) and the auricle at once responds to each stimulus (Fig. 1). It is not, as Engelmann would have had us believe, a rise of excitability which is responsible for this reaction, but an earlier development of the responsive stage, consequent upon the shortening of systole. The vagus depresses all the properties of the muscle which it influences, and apparently simultaneously, but by depressing them it renders the muscle responsive to shocks at an earlier phase of the cycle.

Engelmann used threshold shocks; the phenomenon is equally well displayed when much stronger shocks are used.

Thus Engelmann's most striking evidence for the independence of the properties excitability and contractility in heart muscle must be disallowed; being disallowed, the remaining evidence appears to us insufficient to establish his main contention.

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